A Case of Diabetic Muscle Infarction in Japan

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Diabetic muscle infarction (DMI) is a rare complication of diabetes mellitus. We report the first recorded case in Japan. A 45-year-old Japanese woman presented with severe pain in the left antero-medial thigh. She had a 14-year history of Type 2 diabetes mellitus (DM). She had first noticed pain in her left thigh after a walk 2 weeks prior to presentation. The pain worsened progressively. She noticed a firm mass in her left thigh. T2-weighted magnetic resonance imaging (MRI) demonstrated a high-intensity signal in the muscle bulk of the anterior component of the left thigh. A needle biopsy of the mass showed necrosis. She was treated with bedrest and an antiplatelet agent. The mass disappeared 8 weeks after admission. DMI is a rare complication of poorly controlled diabetes mellitus. Twenty-seven cases with DMI have been reported in the English literature but we believe this is the first Japanese case with DMI. © 1998 John Wiley & Sons, Ltd.

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Introduction

Diabetic muscle infarction (DMI), first described by Angervall and Stener in 1965, is a rare complication of diabetes mellitus.[1] We report the first Japanese case with the permission of the patient.

Case Report

A 45-year-old Japanese woman was admitted to Ryukyu University Hospital because of severe pain in the left antero-medial thigh. She had a 14-year history of Type 2 diabetes mellitus (DM), and had been treated with diet alone. Her body mass index was 18 kg m^{-2}. She had severe diabetic complications: proliferative retinopathy, end-stage diabetic nephropathy, autonomic nerve dysfunction and calcification of her abdominal aorta. She had had gas gangrene in her right lower leg 2 years before admission, when her right lower leg was amputated. She had been well until 2 weeks before admission, when she first noted pain in her left thigh. The pain gradually became worse. She noted swelling on her left thigh 8 days before admission. She had been in bed with severe pain over 2 days before admission. There was no history of preceding trauma.

Physical examination revealed a firm 5 × 3 cm mass with a distinct margin on the left thigh; there was no local heat or skin rash. The power and range of movement of her muscles were markedly limited by pain. Homans' sign was absent.

Examination of the blood showed a white blood cell count of 12.3 × 10^9 l^{-1} (with no shift to the left); a haematocrit of 14.5 %, fasting plasma glucose of 7.5 mmol l^{-1}; serum creatinine level of 627.6 μmol l^{-1}; serum creatine kinase (CK) level of 222 IU l^{-1} (normal<166 IU l^{-1}) and a serum C-reactive protein level of 1.3 × 10^6 μg l^{-1} (normal< 2.8 × 10^4 μg l^{-1}). The glycosylated haemoglobin (HbA_{1c}) was 6.3 % (non-diabetic range < 5.8 %). No pyogenic micro-organisms were detected in cultures of serial blood samples and a local aspirate. T1-weighted magnetic resonance imaging (MRI) on admission demonstrated asymmetry in the muscle bulk of the anterior component of the left thigh. There was no bone destruction or abscess. T2-weighted MRI showed a high-intensity signal in the area involving the left vastus lateralis muscle, the vastus medialis muscle, the vastus intermedius muscle, the rectus femoris muscle, and the sartorius muscle (Figure 1A). A needle biopsy performed on day 5 showed necrosis of muscle fibres, thickening of the fascia with neovascularization, cuffing of the perivascular lymphocytes, interstitial fibrosis, and proliferation of small vessels (Figure 2). DMI was diagnosed. She was treated with bedrest, narcotic analgesics, and an antiplatelet agent. Haemodialysis was also started.

The pain disappeared on day 14. She regained normal thigh mobility on day 20. The mass disappeared completely 8 weeks after admission. A follow-up MRI showed a resolution of the initial findings (Figure 1B). She was discharged after 10 weeks. There has been no recurrence.

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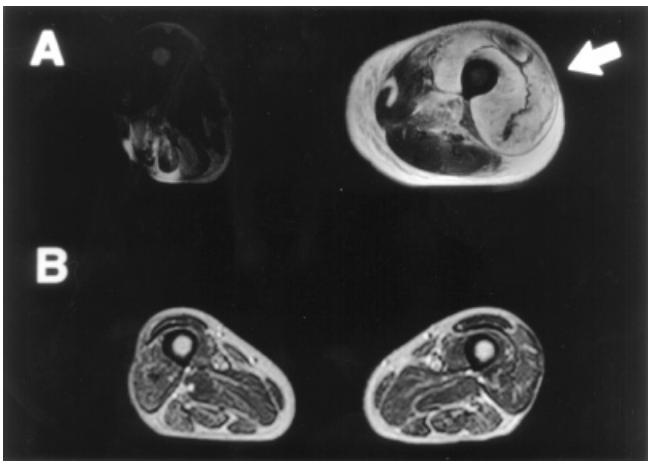

Figure 1. (A) Axial T2-weighted (TR 4500/TE 99) MR image of the left mid-thigh on admission demonstrated an abnormal high-signal area in the vastus lateralis muscle, the vastus medialis, the rectus femoris muscle, the vastus intermedius, and the sartorius muscle (arrow). (B) Axial T2-weighted (TR 3000/TE 104) MR image of the left mid-thigh on day 50 showed disappearance of the abnormal signal

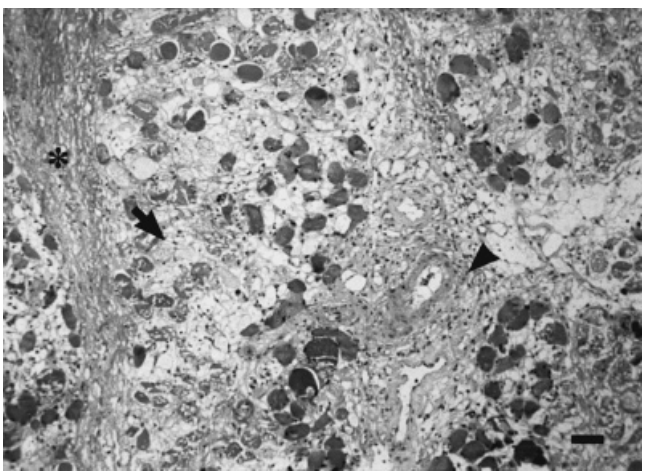

Figure 2. A histological specimen of the quadriceps on day 5 showed a large area of muscle necrosis (arrow), proliferation of connective tissue (asterisk), and hyalinized small vessels (arrowhead); bar = 40 μm; hematoxylin and eosin stain

Discussion

This is the first Japanese case with diabetic muscle infarction (DMI). DMI is a rare complication of poorly controlled diabetes mellitus. Twenty-seven cases with DMI have been reported in the English literature but we could find no Japanese patient with DMI.[1–9] The mean age of the 27 reported cases was 39 ± 12 years and the duration of diabetes mellitus ranged from 25 to 65 years. Most of the patients had severe and multiple microvascular complications. Our patient had evidence of reasonable blood glucose and HbA_{1c} on admission. However, she had multiple severe diabetic complications and it is possible that her blood glucose levels had been high before.

The clinical features of DMI are a sudden onset of pain and swelling of the thigh or calf muscle without systemic symptoms or signs indicative of infection. After several days, a firm painful mass appears without skin infection. Spontaneous resolution occurs over a period of several weeks to months of bedrest. Although CK levels remain near or within the normal range in most cases, they sometimes increase during the first few days.[3] MRI findings are low-intensity signals on T1-weighted images and high-intensity signals on T2-weighted images in the infarcted muscles.[2–9] Histological features include a large area of muscle necrosis with connective tissue proliferation and small vessel occlusion. In the acute phase, necrosis of the muscle and nerve with infiltration of polymorphonuclear cells is seen. In the subacute phase, proliferation of connective tissue and embryonal muscle cells is pronounced. The proliferation explains the mass. In the chronic phase, small blood vessels become recanalized; the necrotic tissue is replaced by new muscle fibres, and the mass disappears.[3] Occlusion of small vessels may play an important role in the development of DMI.

The differential diagnosis includes: primary muscle tumour, focal nodular myositis, abscess, and thrombophlebitis. Muscle biopsy is useful in the acute phase, because MRI findings of necrotic tissue and inflammation are similar. In the management, we consider that thrombolytic therapy will be useful to reduce the infarcted area in the acute phase, although patients with DMI have previously been treated with bedrest and analgesics. Antiplatelet agents may also be useful to prevent recurrence. Bjornskov *et al.* have suggested that patients with DMI were in a hypercoagulated state and proposed the use of warfarin therapy to prevent recurrence.[8] Our patient was treated with aspirin and has not experienced recurrences.

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